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IMPACT OF CHANGES IN BLOOD PRESSURE DURING THE TREATMENT OF ACUTE DECOMPENSATED HEART FAILURE ON RENAL AND CLINICAL OUTCOMES: AN APPLICATION OF THE ESCAPE TRIAL LIMITED DATASET

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Monday, April 04, 2011, 9:30 a.m.-10:45 a.m.

Session Title: Cardiomyopathies/Myocarditis/Pericardial Disease
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Background: In addition to vascular resistance, the primary determinate of blood flow in regional vascular beds is perfusion pressure. The major goal of this study was to evaluate if reduction in blood pressure during treatment of decompensated heart failure was associated with worsening renal function (WRF) and if this potentially treatment induced form of WRF was associated with an adverse prognosis.

Methods: Subjects in the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness trial limited data set with baseline/discharge systolic blood pressure (SBP) and creatinine levels were studied (386 patients).

Results: Reduction in SBP was greater in patients experiencing WRF (-10.3 ± 18.5 mmHg vs. -2.8 ± 16.0 mmHg, $p<0.001$) with larger reductions associated with greater odds for WRF (OR=1.3 per 10 mmHg reduction, $p<0.001$). A reduction in SBP above the median was associated with greater doses of in-hospital oral vasodiolators ($p</=0.017$), thiazide diuretic use ($p=0.035$), and greater weight reduction ($p=0.023$). In patients with a reduction in SBP above the median, WRF was not associated with increased mortality (adjusted HR=0.76, $p=0.58$). However, in patients with a reduction in SBP below the median, WRF was strongly associated with increased mortality (adjusted HR=5.3, $p<0.001$, p interaction=0.001).

Conclusions: Reduction in blood pressure during the treatment of decompensated heart failure is strongly associated with WRF. In the setting of a significant reduction in blood pressure, WRF does not appear to affect prognosis, whereas WRF in the absence of a significant lowering in blood pressure is strongly associated with increased mortality. These observations support the notion that WRF may be the result of several different cardio-renal phenotypes with potentially dissimilar effects on outcomes.